Articles

Occupational Asthma
Practical Points for Diagnosis and Management

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Asthma is a common chronic illness characterized by episodes of reversible airflow obstruction. A cornerstone of asthma management is identifying and avoiding agents that cause bronchospasm. The workplace is an important potential source of respirable exposures that can cause or trigger asthma. Identification of an occupational factor in asthma is important: early diagnosis and removal of the worker from the exposure is associated with improved prognosis; the diagnosis of occupational asthma may lead to compensation for work-related impairment and disability; and the diagnosis of occupational asthma is a Sentinel Health Event with implications for public health and prevention. In this article, we review specific causes of occupational asthma and general settings in which an occupational factor should be suspected and explored as part of the management of the worker with asthma. We also review specific and simple elements of history and pulmonary function testing that can be easily assessed by most health care practitioners and may be sufficient to establish a diagnosis of occupational asthma. Finally, we review the medical-legal implications of occupational asthma.

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Asthma is a common chronic inflammatory disease affecting the airways. It is characterized by episodic airflow obstruction that typically reverses either spontaneously or with treatment. Signs and symptoms include wheezing, cough, chest tightness, and shortness of breath. Asthma is both prevalent and costly. An estimated 15 million people are affected in the United States, including approximately 10 million adults.[1] The total costs of asthma in the United States alone are in excess of $6 billion, including $2.6 billion due to lost work.[2]

The proportion of adult cases of asthma with occupational factors has been estimated at between 5% and 20%.[3,4] Of those diagnosed with occupational asthma, 20% to almost 50% may suffer a reduction in income after the diagnosis is made.[5,6] Work disability resulting in job change was observed in 20% of adults with asthma in one study over a 5-year period.[7] Despite its important adverse impact on health and work, occupational asthma is likely underdiagnosed; even when the diagnosis is suspected, problem management and follow-up is inadequate in as many as half of cases.[8]

Occupational asthma can be defined broadly or narrowly. Broadly defined, occupational asthma is reversible airflow obstruction that is either caused by or exacerbated by a workplace exposure. This very inclusive definition includes cases in which a diagnosis of asthma may antedate a workplace exposure suspected of provoking bronchospasm. In such cases, an occupational exposure may be identified as an important *trigger* of asthma, but it would not be the *cause* of the disease per se. For example, a susceptible food handler who develops bronchospasm when exposed to the refrigerated air of an industrial cold room would, under this definition, have occupational asthma despite the fact that cold air may, of course, be encountered outside of the workplace and despite the fact that cold air exposure is not a cause of asthma per se.

More narrowly defined, occupational asthma includes only those cases of asthma that result from an exposure encountered exclusively at work. In this descriptive model of occupational asthma, a workplace exposure is etiologic; that is, it is the principal *cause* of asthma in the affected worker. In other words, if not for the occupational exposure, the individual would not have asthma. To satisfy this strict case definition, asthma must be newly diagnosed and temporally linked with a workplace exposure that has, either historically or experimentally, been demonstrated to cause asthma in previously healthy workers. An example of this would be new-onset asthma in a health care worker who has had daily exposure to glutaraldehyde since being hired several months previously as an endoscopy technician.[9] In contrast, persons with preexisting asthma who report

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ABBREVIATIONS USED IN TEXT

RADS = reactive airways dysfunction syndrome
MSDS = Material Safety Data Sheet
FEV_1 = forced expiratory volume in 1 second
TDI = toluene diisocyante
Ig = immunoglobulin
NIOSH = National Institute for Occupational Safety and Health
SENSOR = Sentinel Event Notification System for Occupational Risks

episodes of bronchospasm attributable to nonspecific workplace irritants such as nuisance dust would not fulfill this case-definition of occupational asthma.

While strict operational case definitions of occupational asthma are crucial in epidemiological investigations, they are less relevant in clinical practice. In terms of disease management and particularly with respect to prevention strategies, the more inclusive description of occupational asthma should be adopted. An alternative term for broadly defined occupational asthma is "work-related asthma." Again, this term would include both work-related aggravation of preexisting asthma as well as new-onset asthma caused by a workplace exposure.

An inclusive definition of occupational asthma has been used and advocated by several investigators. Newman-Taylor[10] and subsequently Cartier[11] defined occupational asthma as "variable airways narrowing causally related to exposure in the working environment to airborne dusts, gases, vapours, or fumes." More recently, Wagner and Wegman advocated that occupational asthma be defined as "all asthma caused or exacerbated by workplace exposures—including asthma from sensitizers, irritant-induced asthma, and workplace exposure–induced asthma attacks."[12] In a recent descriptive study of occupational asthma, Wheeler et al defined occupational asthma as a condition meeting the following criteria:

> a patient has asthma and an association between symptoms and the workplace, either documented with specific testing or by clinical history; there has been a workplace exposure, with an association between asthma symptoms and exposure to some process, substance, or environment at work; there are work-related changes in spirometry or peak flow results; or there is a positive response to bronchial provocation testing with the agent to which the patient was exposed at work.[13]

Work-aggravated asthma may be compensable under workers' compensation law. In one recent investigation, Tarlo et al reviewed 609 claims of occupational asthma to a workers' compensation board,[14] of which 469 were found to be associated with workplace exposures. Of those, 50% (or a total of 39% of all claims filed) were cases of asthma aggravated by irritant workplace exposures, while 50% were cases of asthma determined to be caused by the workplace, typically by a known sensitizer such as isocyantes. Importantly, compensation was available for both work-aggravated asthma and asthma thought to be caused by a known sensitizer from the workplace.[14] Additionally, Harber[15] has described a variety of occupation–asthma interactions that should be used to characterize the effect of the workplace on asthma and are relevant to disability reports filed for occupational asthma claimants; included among these is work aggravation of preexisting asthma.

Although clinicians should generally adopt an inclusive view of occupational asthma, it is nonetheless useful to make distinctions between the various types of workplace exposures that may contribute to asthma to develop a more complete understanding of clinical presentation and illness natural history. In considering all those with asthma, a remarkably heterogeneous constellation of factors play roles in disease expression, ranging from emotional to genetic. The spectrum of factors that commonly receive consideration in the evaluation of occupational asthma, however, is more circumscribed and generally limited to inhaled airway stimuli. Still, this represents a very large group; more than 200 occupational agents have been causally linked with asthma.[16] For the purposes of this review, we distinguish between exposures that have been demonstrated to cause asthma, including immune sensitizers, and exposures that aggravate or trigger preexisting asthma.

Occupational Causes of Asthma

The major classifications of occupational exposures that cause new-onset asthma are *1*) high-molecular-weight compounds; *2*) low-molecular-weight compounds; and *3*) high-level-irritant inhalant exposures. Importantly, those who develop asthma after exposure to these inhalants acquire a chronic disease that they presumably would never have developed had they not been exposed. Among susceptible individuals, continued exposure to these inhalants can be expected to result in progressive worsening of asthma.[17]

High-molecular-weight organic compounds include plant and animal proteins, vegetable gums, and wood dusts or barks. Many of these substances induce specific immunoglobulin E (IgE) antibodies, biomarkers of sensitization, in the susceptible worker. Table 1 lists examples of occupations that expose workers to high-molecular-weight compounds that have been causally linked with asthma. A more comprehensive listing may be found elsewhere.[18] Among some exposed workers, asthma symptoms may persist even years after removal from these exposures.[19]

Low-molecular-weight, mostly inorganic, compounds that cause occupational asthma include a variety of chemicals, include isocyanates, which are used to make polyurethane plastics, and metals, drugs, and other chemicals.

One of the most extensively investigated low-molecular-weight substances demonstrated to induce asthma is plicatic acid (440 Da), the primary compound in the Western red cedar and the principal etiologic factor in cedar asthma. Exposure to Western red cedar and, in turn,

TABLE 1.—*Selected workers at risk for occupational asthma caused by high-molecular-weight compounds*

Occupations	*Etiologic exposure*
Farmers	Grain dust; grain mites
Printers	Vegetable gums
Health care workers	Latex
Textile workers	Wool
Bakers	Plant dust and fungal enzymes
Animal handlers	Urine and dander
Woodworkers/sawmill workers	Wood dusts
Postal workers/Bookbinders	Glues

TABLE 2.—*Selected workers at risk for occupational asthma caused by low-molecular-weight compounds*

Occupations	*Etiologic exposure*
Roofers/insulators	Toluene diisocyante
Paint/plastic/chemical workers	Trimellitic anhydride
Pharmaceutical workers	Antibiotics
Health care technicians	Formaldehyde, glutaraldehyde
Printers	Chromium
Textile workers	Dyes
Hard metal grinders, carpenters (construction/woodworking)	Cobalt plicatic acid (Western red cedar)

plicatic acid occurs in a variety of occupations including sawmill work, carpentry, and construction.[20–24] Another important group of low-molecular-weight compounds is the acid anhydrides. These substances can cause symptoms of immediate airway irritation as well as asthma. Trimellitic anhydryde is among the more important, widely encountered acid anhydrides in industry. Exposure to trimellitic anhydryde occurs in the production of epoxy and alkyd resins used to manufacture coating materials such as paints and plastics.[25–27] Examples of some important occupational exposures to low-molecular-weight agents that cause asthma are shown in Table 2. A more comprehensive listing may be found elsewhere.[18]

A number of findings support the concept that immunologic mechanisms play an important role in mediating asthma caused by both low- and high-molecular-weight compounds. Evidence supporting a role for the immune system includes the observation that an immunoglobulin response may be found following exposure to many of the high-molecular-weight compounds that cause asthma. Although not typical of the low-molecular-weight compounds, some of these inducers of asthma such as the acid anhydrides also promote immunoglobulin responses. Importantly, the agents implicated as etiologic in occupational asthma do not cause disease in all individuals, again suggesting that host response is an important determinant in shaping the natural history in the exposed worker. Additionally, a period of latency that may last for weeks, months, or years can be expected between initial exposure and evolution of frank asthma. Finally, once the affected individual is sensitized from initial exposures (that is, the immune system is primed), extremely low concentrations of the substance can provoke subsequent episodes of bronchospasm. For example, in the sensitized individual, toluene diisocyanate (TDI) can induce bronchospasm in concentrations as low as one part per billion.[28]

These observations underscore two very important and clinically relevant points regarding the diagnosis and management of occupational asthma. First, the latency between initial exposure and onset of symptoms means that the concept of a "new" workplace exposure must be interpreted broadly. Second, the potential for an exceptionally low-dose exposure to precipitate bronchospasm among sensitized persons typically renders personal respirators ineffective in preventing exacerbations. Instead, complete avoidance of the exposure should be encouraged, which will typically necessitate a job change. Other options that should be considered, whenever possible, include substituting a nonsensitizing material for the suspected asthma inducer or engineering controls to reduce the risk of exposure. These interventions may prevent the affected worker from having to make a major job change. They may also have the benefit of eliminating or reducing the risk of new cases of occupational asthma among other workers who are not yet symptomatic. Importantly, occupational asthma should not be viewed as an idiosyncratic response to a workplace exposure. Instead, a well-defined case of occupational asthma should be viewed as evidence of a potential health hazard in the workplace that requires modification to prevent more cases in the future.

In contrast with agents that appear to require immune system participation to cause asthma, irritant inhalant exposure in high concentrations can cause asthma through apparently nonimmunological mechanisms. Short-term, high-level exposure to irritant fumes, vapors, or smoke can cause reactive airways dysfunction syndrome (RADS). RADS refers to asthma that is caused by a single toxic inhalational exposure.[29–31] The key feature of RADS is that, by definition, the affected individual must have had a documented high-level irritant exposure, usually resulting from a single major industrial accident. Symptoms typically develop within hours of the exposure. Strictly defined, the affected individual may not have had a preexisting diagnosis of asthma or a syndrome of reversible airflow obstruction. As in other forms of asthma, exacerbations and remissions can be expected in RADS, although most people with RADS have less reversibility in their airflow obstruction compared with others with asthma.[32,33]

Occupational Triggers of Preexisting Asthma

A wide variety of occupational exposures can trigger bronchospasm among persons with preexisting asthma.

TABLE 3.—*Settings in which to suspect an occupational factor in asthma*

1. All cases of adult-onset asthma
2. Cases in which a worker suggests a link between the workplace and deterioration in respiratory health
3. Asthma that began or worsened after a job change
4. Asthma that follows a one-time high-level irritant inhalant exposure (for instance, Reactive Airways Dysfunction Syndrome)
5. Occupations with airborne exposures that can be easily seen or smelled (dust, vapors, smoke)
6. Occupations in which the worker is advised to use a personal respirator

An exposure that may trigger bronchospasm in one individual, however, may have no significant effect on airway function in others with asthma. Moreover, the intensity and duration of bronchospasm triggered by a given provocative exposure will vary among persons with asthma in ways that are not easy to explain or predict.

Gases, dust, smoke, and particles have all been shown to cause airflow limitation in people with asthma.[34] Chlorine gas is the most common irritant gas exposure encountered in industry. Workers in textile, pulp bleaching, and water purification industries may be affected.[35] Dust can be encountered in any industry that involves movement, disruption, or disintegration of either organic or inorganic materials. Dusty occupations include farming, construction, mining, and certain manufacturing procedures. Smoke is a complex mixture of gases and particulates that results from incomplete combustion. Smoke inhalation resulting from structural fires is a well-recognized cause of airflow obstruction and an obvious occupational hazard among firefighters.[36] Cigarettes are another important source of smoke exposure. Although restrictions on smoking in the workplace are becoming more widespread, they have not been universally adopted. Consequently, secondhand cigarette smoke remains a relevant noxious inhalant, particularly among indoor workers such as restaurant and bar workers and some flight attendants. Finally, inhalational exposure of very small particles in the micron and submicron range has been correlated with reductions in airflow in people with asthma.[37,38] While epidemiological investigations studying respiratory effects of particle inhalation have focused predominantly on ambient air pollutants, they do support the hypothesis that particle inhalation—whatever the source and whatever the chemical composition—may have a deleterious effect on airway function among those with asthma. That is, respirable particles may act in a nonspecific or generic manner to cause bronchospasm.

When To Consider a Diagnosis of Occupational Asthma

An occupational history should be part of the initial evaluation of all adults with asthma, including a complete work history detailing all jobs as far back in time as possible. It is important to recognize that some occupational exposures can be important, but unrecognized, triggers of bronchospasm in people who report having had asthma all their lives. In these circumstances it is easy to dismiss deterioration in respiratory function as unavoidable disease progression when, in fact, deterioration in respiratory function may be attributable to an avoidable workplace exposure. While occupational factors should be assessed in all workers with asthma, several general circumstances, delineated in Table 3, merit particularly close scrutiny. When a worker with asthma reports that one or more of these circumstances is present, the health care practitioner should suspect an occupational factor and pursue further questioning.

How to Diagnose Occupational Asthma

It is axiomatic that a diagnosis of asthma must be established before a diagnosis of occupational asthma can be considered. Moderate and severe bronchospasm is generally readily recognizable and, in those cases, the diagnosis of asthma should be established without difficulty. In cases of mild asthma, including many cases of mild, new-onset occupational asthma, disease presentation can be subtle and the diagnosis may be more elusive. Episodic dry cough, chest tightness, and increased breathing effort may be the only manifestations of asthma in some individuals. Patients may absolutely deny a history of frank shortness of breath or wheezing. Objective data from spirometry and peak flow meter recordings demonstrating airflow obstruction that reverses after the administration of a bronchodilator, or symptomatic improvement after empiric treatment with a bronchodilator, may suggest the diagnosis. A history of spontaneous improvement in respiratory symptoms or function will support a diagnosis of asthma. Additionally, a trial of avoidance to exposures such as dust, smoke, vapors, fumes, and aeroallergens followed by improvement in respiratory status is also strongly suggestive of asthma.

Confirming a diagnosis of occupational asthma and implicating a specific exposure can be challenging. No single test clinches the diagnosis. Since asthma is characterized by exacerbations and remissions, evaluation of a patient's disorder at one point in time may not fully characterize the disorder. Multiple exposures may be relevant, both in and out of the workplace, further complicating the evaluation. Exposure–response intervals that last many hours may complicate recognition of occupational asthma. Specific airway challenges with suspected agents in an exposure laboratory, although appealing in principle, are not widely available or validated. Moreover, they are complicated and cannot fully replicate real-world exposure settings.[39] As a consequence, their use remains largely confined to research. In the end, a preponderance of evidence must be viewed as the clinical gold standard for the diagnosis of occupational asthma in most clinical practices. The diagnosis will generally be established from a detailed history supported by serial lung function studies.

TABLE 4.—*Questions to determine links between the workplace and asthma symptoms*

1. Is there a diurnal temporal relationship between work and the onset of bronchospasm? In other words, do symptoms develop in a predictable manner, either during or after exposures at work or after returning home from work on days with exposure?
2. Do symptoms improve during vacations, over the weekend, or during any other extended period away from work?
3. Are symptoms worse at the end of the workweek?

TABLE 5.—*Questions to assess the job process*

1. In as much detail as you can provide, what do you do?
2. Do you have a Material Safety Data Sheet in your workplace? (If yes, obtain a copy.)
3. Can you see particles or dust in the air at work?
4. Do you blow dust out of your nose or cough up dust at the end of the workday?
5. Are you exposed to vapors, fumes, mists, or chemicals?
6. Are you advised to wear a personal respirator? Have you been personally fitted for one? Do you wear it faithfully?
7. (With worker concurrence only) To the employer or employer safety and health personnel: Please provide any information about industrial hygiene surveys, workplace illness surveillance and monitoring, the presence of other affected workers (current or past), and the presence of any known asthma sensitizers or irritants in the workplace.

Since occupational asthma is, in the end, a clinical diagnosis, the accuracy of the diagnosis and the efficacy of the management that follows from the diagnosis will depend heavily on the quality and availability of relevant data. Moreover, the consequences of a false-positive versus a false-negative diagnosis of occupational asthma for a given worker will necessarily have an impact on data interpretation and, accordingly, the management of that worker. Inevitably, real-world issues, including the economic consequences to the patient and the employer, will influence prevention and treatment strategies.

At a minimum, the history should address each of the six special circumstances detailed in Table 3. Other important questions which address the temporal relationship between symptoms and work may help to establish an exposure–response relationship between the workplace and bronchospasm (Table 4). Even though these classic exposure–response temporal relationships are often present early in the course of illness, they may not be apparent later in the course, thereby complicating diagnosis.

It is important to note that specific preformed IgE bound to mast cells mediates asthma caused by many high-molecular-weight compounds. Exposure to these high-molecular-weight compounds can, in the sensitized person, provoke an immediate response, or early-onset asthma. Peak symptoms will often occur within 30 minutes of exposure.[3] In contrast, low-molecular-weight compounds may cause late-onset occupational asthma symptoms that may not develop until 4–6 hours after exposure, with peak symptoms developing 8–10 hours after exposure.[3] Finally, both low- and high-molecular-weight compounds can cause a dual response, with features of both early- and late-onset asthma. In any case, diurnal patterns of bronchospasm related to workplace exposure should suggest occupational asthma.

Details about the job process may suggest relevant occupational exposures. A job title, by itself, rarely provides a complete picture of the work task. A series of questions (listed in Table 5) can provide circumstantial evidence linking the workplace with respiratory symptoms. The worker should be instructed to obtain information about hazardous materials in the workplace. The Occupational Safety and Health Administration requires employers to make Material Safety Data Sheets (MSDS) readily available to workers. These documents provide a detailed list of hazardous materials in the workplace and their potential toxicities, including respiratory effects. The Chemical Manufacturers' Association CHEMTREC can also provide assistance in obtaining chemical specific information (1-800-262-8200). MSDSs should not be viewed as the sole reference for possible exposures that can cause or trigger asthma. With the approval of the worker, communication with the employer regarding workplace materials and existing environmental surveys can be an additional source of information regarding possible important exposures.

Another valuable resource is the National Institute for Occupational Safety and Health (NIOSH) which provides, by telephone or Internet, a wide variety of information on worker health, including occupational respiratory disorders (1-800-356-4674; http://www.cdc.gov/niosh/homepage.html). Exposure assessment and diagnostic strategies have also been described in a recent consensus statement published by the American College of Chest Physicians.[40]

There are no physical examination findings that are specific for occupational asthma. Skin testing may establish whether an individual is sensitized to a specific antigen. A substance that produces a positive wheal and flare on skin testing, however, may not necessarily be a cause or trigger of asthma in a given worker. Further, immunologic testing is, with a few exceptions, useless in testing for sensitization to low-molecular-weight compounds.

After the history and physical examination, repeated pulmonary function testing is the most important tool in diagnosing occupational asthma. Any objective data about lung function predating a work exposure suspected of causing or exacerbating asthma can provide valuable baseline data. Typically, however, in the previously asymptomatic individual, lung function studies will never have been performed. Serial peak expiratory flow monitoring with a handheld ambulatory meter or spirometry demonstrating changes in airflow temporally related to work support a diagnosis of occupational asthma. Examples of peak expiratory flow rate monitors are shown in Fig. 1. Diurnal variability of 20% or greater in peak flow

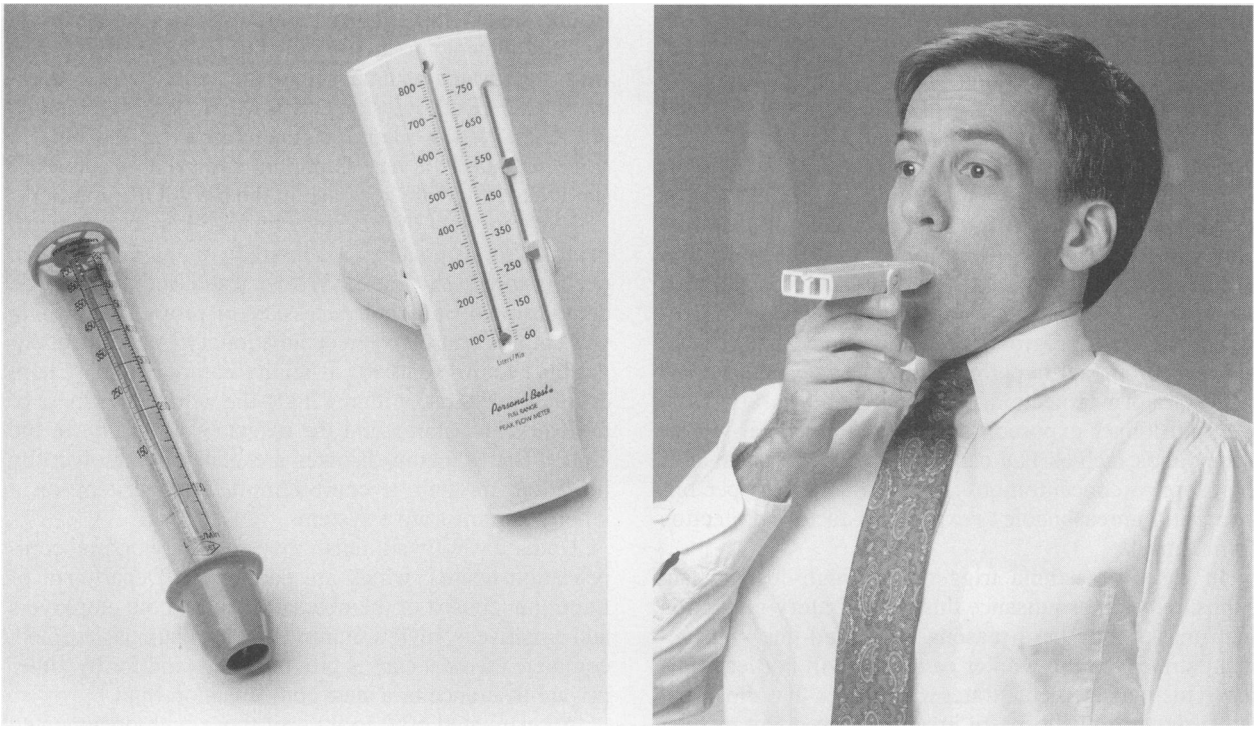

Figure 1.—Ambulatory peak expiratory flow rate monitoring. Measurement of peak expiratory flow rate every 2 hours for a 2-week period at work and away from work provides objective data that can support a diagnosis of occupational asthma. A. Examples of peak flow meters: TruZone, Monaghan Medical Corp (Plattsburgh, NY) (left) and Personal Best, HealthScan Products, (Cedar Grove, NJ) (right). B. Proper technique for measuring peak expiratory flow: upright position; firm seal around the mouthpiece; lungs filled completely at the beginning of the maneuver. The forced expiratory maneuver is performed as hard and fast as possible. The greatest of three consecutive maneuvers is recorded.

indicates abnormal changes in airflow over short periods of time consistent with asthma.[41] Peak expiratory flows should be recorded every 2 hours while awake on work days and ideally for a 2-week period away from work.[39,40,42,43] Importantly, in those individuals who demonstrate a late asthmatic response, decrements in lung function may be most pronounced hours after they have left the workplace. Documentation of clear improvement in lung function after prolonged absence from the workplace, such as after vacations, is very strong evidence for an occupational exposure factor.

Occupational asthma ultimately remains a clinical diagnosis. The diagnosis is supported by a focused evaluation that should establish a history of workplace exposure to a recognized noxious agent. Temporal links between a relevant occupational exposure and both asthma symptoms and airflow obstruction provide strong additional support for the diagnosis.

Prevention, Treatment, and Legal Implications

Prevention is the cornerstone of management of occupational asthma. If a preponderance of evidence supports a link between the workplace and asthma, then measures should be advocated to modify the worksite or remove the worker from the exposure setting. The primary effort should be to modify the workplace to prevent continued exposure. Since one case of occupational asthma may presage others, workplace modifications may have beneficial effects that extend beyond the symptomatic worker. Specifically, workplace modifications may prevent the evolution of illness in other workers who have not yet experienced symptoms or significant loss of lung function. In some cases, however, the worker will need to change job tasks. It is the obligation of physicians to report cases of occupational asthma to state and/or county health boards under the OSHA act of 1970. More information on industrial hygiene issues can be found at the OSHA web site (http://www.osha.gov/wutsnew.html).

Early removal of the worker from the suspected exposure has important prognostic implications. Multiple studies have demonstrated that asthma symptoms linked to a workplace exposure may persist years after removal from the exposure.[44–46] Studies have also shown that early diagnosis of occupational asthma and early removal from an inciting exposure are important in regaining lung function and controlling symptoms.[47–50]

Usual asthma therapies may be used in managing asthma resulting from occupational exposures. These include beta-2–specific adrenergic agonists, corticosteroids, and adjuvant anti-inflammatory medications. A stepwise approach to the use of asthma medications and to

asthma management was well delineated in a monograph recently produced by the National Institutes of Health National Asthma Education and Prevention Program.[41] Bronchodilators and other asthma medications should never substitute for preventive measures, however.

The use of masks to provide respiratory protection is usually not an effective intervention and should not be viewed as a substitute for workplace modification or removal of the worker from the exposure setting. For example, among workers with TDI-induced asthma, continued occasional exposure to TDI has been demonstrated to cause progressive deterioration in lung function and bronchial hyperresponsiveness despite the use of respiratory protection compared with workers who were no longer exposed.[51] Since low-molecular-weight compounds such as TDI can induce bronchospasm after exposure to concentrations as low as one part per billion,[28] it is unreasonable to expect satisfactory protection from a mask.

In cases of asthma triggered by high-dose irritant exposure, such as nuisance dusts, respiratory protection with masks may be a reasonable second-line management strategy if the worker cannot or will not leave the job. This management strategy presumes that most irritants trigger bronchospasm in a dose-dependent manner through nonallergic mechanisms. Such a dose-response relationship has been reported among boilermakers exposed to fuel ash, a particulate generated from fossil fuel combustion. Hauser et al[52] reported the results of a prospective evaluation in which they showed a significant dose-response relationship between the decline in the adjusted change in airflow and the peak and average particulate exposure during a 4-week period. Nevertheless, despite the fact that a reduction in nuisance dust and irritant exposures afforded by respiratory protection masks may have a salutary health effect among people with asthma, complete avoidance of triggers should remain the principle management goal.

Disease surveillance is an essential component in the prevention of occupational asthma. In a recent discussion of strategies to prevent occupational asthma, Venables[53] highlighted the importance of surveillance to gain information on how common asthma is relative to other occupational lung diseases and on the relative frequency with which different agents cause asthma. Occupational safety and health is one of the priority areas targeted in the US Department of Health and Human Services report, "Healthy People 2000."[54] In the United States, the Sentinel Event Notification System for Occupational Risks (SENSOR), in collaboration with NIOSH, has been developed as a national program for occupational disease surveillance, including occupational asthma.[55] The program involves collaboration between NIOSH and state and county health departments. The surveillance case definition used by NIOSH is inclusive and includes all cases of asthma caused by or aggravated by workplace exposures.[56] A classification scheme using the overarching term "work-related asthma" has been proposed as part of planned report of findings from SENSOR-participating states. Surveillance case definitions are currently in development for this report, but both new-onset and work-aggravated asthma will be included (written communication; Ruth Jajosky, DMD, MPH; NIOSH). Finally, physician-filed workers' compensation reports are, in some US states, a very important and reliable source of surveillance and are accessed in the SENSOR program.

It is important to recognize that workers can be compensated for asthma that is caused by or aggravated by an occupational exposure. While impairment must be demonstrated, it is not necessary to prove that asthma was entirely caused by an occupational factor to meet eligibility requirements for disability compensation. Claim disputes may arise with regard to the work-relatedness of the worker's asthma and the extent of disability. In the United States, claim disputes are generally resolved by litigation through a court-administered system or a wholly administrative system.

Under a wholly administrative system, workers' compensation boards, which are part of the Department of Labor but consist of members chosen by both employers and employees, review claims. When a claim is accepted, complete medical care is provided and paid for by either private insurance or a state compensation fund.[57]

An estimated 60% to 90% of those with documented occupational asthma continue to demonstrate respiratory impairment after leaving an exposure.[58] Accordingly, workers may be entitled to compensation for work-related disability. Disability is different from impairment. Impairment is defined as a functional abnormality resulting from a medical condition. It may be temporary or permanent. Disability indicates the total effect of impairment on the individual and will depend on the job and the worker's ability to compete in an open job market. Impairment is determined by the physician, and disability is determined by disability raters, workers' compensation judges, and review boards.

Guidelines for determining impairment due to asthma have been developed by the American Thoracic Society.[59] The guidelines use a scoring system consisting of several physiologic and clinical parameters to characterize impairment: *1*) postbronchodilator forced expiratory volume in 1 second (FEV_1) measured by spirometry, *2*) reversibility of FEV_1 or degree of airway hyperresponsiveness, and *3*) minimum asthma medication need. Recovery from occupational asthma may continue for as long as 2 years after removal from an exposure.[19] Accordingly, assessment for long-term impairment and disability should be carried out 2 years after the removal from an exposure, at which time improvement can be expected to have reached a plateau.

In summary, occupational asthma is a prevalent disorder caused by a wide variety of respirable exposures including organic and inorganic compounds. A diagnosis of occupational asthma will only be established if there is suspicion that asthma symptoms may be causally linked to the workplace. The diagnosis is clinically based and is supported by evidence gained from a detailed and focused history and pulmonary function

testing, including ambulatory peak expiratory flow monitoring. Material Safety Data Sheets and other sources provide worksite-specific information about hazardous exposures and may be useful in identifying a specific etiologic exposure. The cornerstone of management is prevention, which typically mandates a change in the work environment or significant work task modifications. Workers with occupational asthma are entitled to compensation, including disability compensation for workers with evidence of persistent impairment after removal from the workplace exposure. Claims disputes are usually settled through litigation.

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